Stenting Both before and after Coiling of a Ruptured Fusiform Basilar Aneurysm to Overcome Mechanical Occlusion Caused by Thrombosis within the Coil Mass

A Case Report

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Summary

Tortuous vessel anatomy reconstructed by stent-assisted coiling may be kinked by thrombus expansion within the coil mass. This can be overcome by placement of a second stent.

Introduction

Fusiform intradural aneurysms of the posterior fossa presenting with hemorrhage are likely dissecting in origin [1-3]. These have high rebleeding rates of up to 85% in the acute phase [4,5]. Those aneurysms located at the basilar artery or vertebrobasilar junction have a worse outcome than those of the intradural vertebal alone or of the posterior cerebral arteries, and may be a separate disease entity [1]. Hypertension and atherosclerosis may be a causative factor [5,6].

This location presents with a greater chance of neurological complication, 44% mortality by 33 months and a mean GOS score at long-term followup of 4.01. Treatment with stent-assisted coiling is necessary if there is no collateral supply to the distal vasculature and is faster to institute and possibly more effective than flow reversal after a bypass. Surgical treatment of large aneurysms of the basilar trunk seems to be difficult and associated with poor outcome [7]. Coiling without a stent is difficult and commonly requires some degree of parent vessel sacrifice, not possible in this case [8]. As poor outcome has been shown to be related to residual filling1, stent-assisted coiling may hold an advantage over reconstruction of the parent vasculature with flow-deflecting stents alone. These aneurysms are characterized by tortuosity [2], as in this case with a focal 90 degree curvature (Figure 1). Tortuosity has been associated with increased wall shear stress and chance for aneurysm growth and hemorrhage [9]. This paper makes the case that this tortuosity may also represent an unexpected impediment to successful treatment that at least in this case was overcome by placement of a second stent.

Case Report

History

The patient was a 55-year-old right-handed man who suffered a headache followed by seizures and syncope during intercourse at 12:00 on 21st October 2008. On arrival at the ER he was found to have 7/10 headache. CT showed fourth ventricular blood, a small amount of subarachnoid hemorrhage within the posterior fossa, and a left-sided prepontine mass with calcification suggestive of an atherosclerotic and possibly dissecting aneurysm (Figure 2). CT angiography disclosed a fusiform aneurysmal dilatation of a tortuous basilar trunk (Figures 1,3,4). There were no significant posterior

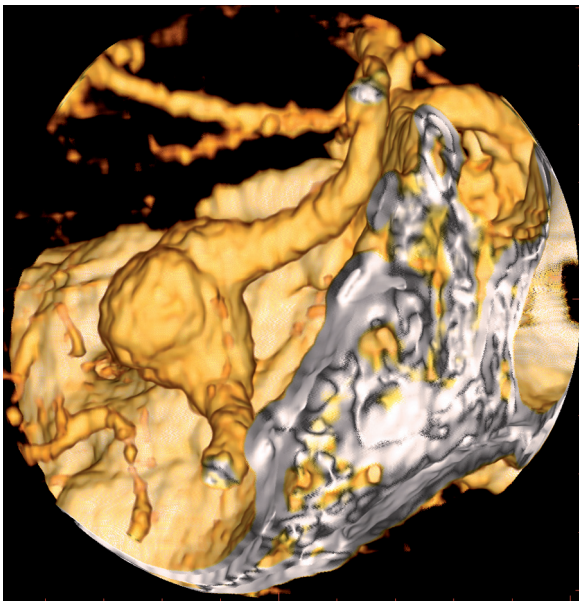

Figure 1

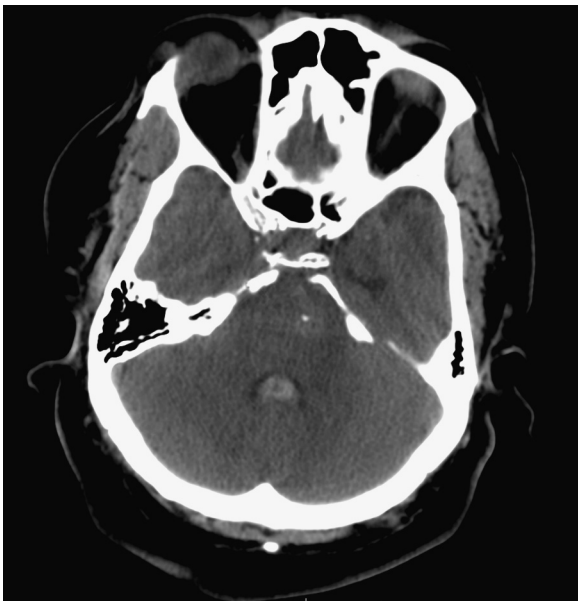

Figure 2

communicating arteries and the smaller left vertebral artery appeared to end in PICA. The patient was on 81 mg ASA and 75 mg Plavix up to the day before ictus because of coronary stenting performed last in November 2005. Remote medical history was notable for central obesity, sleep apnea, hypercholesterolemia, hypertension, diabetes, coronary artery disease, prior CHF and decreased renal function. Remote surgical history was notable for coronary artery stenting x 5. Family history was negative for stroke or intracranial hemorrhage. Social history was notable for smoking of approximately one pack a day for the last five years. Medications included Nitropaste, Norvasc, CoReg, TriCor, and Lipitor in addition to the

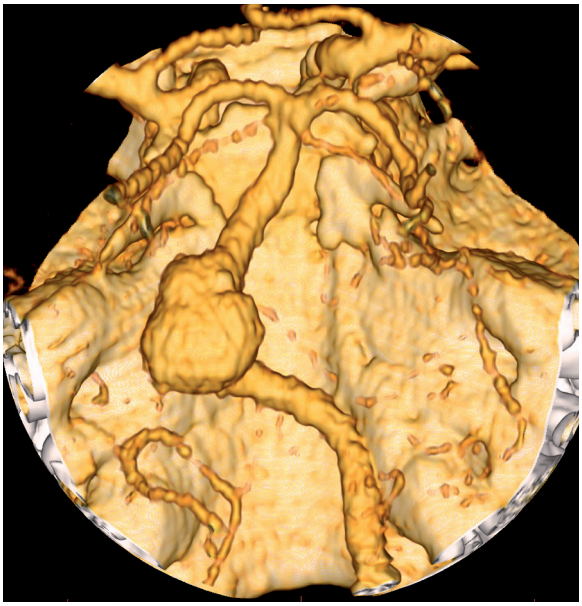

Figure 3

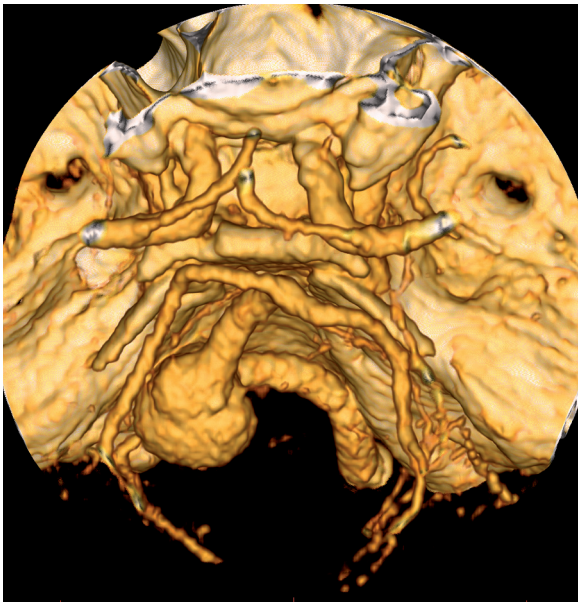

Figure 4

ASA and Plavix noted above. Physical examination was of an obsese man with a headache and left-sided hemianesthesia sparing the face. Laboratory tests were notable for a glucose of 132 and creatinine of 1.7. He was hydrated overnight and his creatinine decreased to 1.5 by the time of angiography and treatment the next morning.

Procedure

Angiography confirmed lack of collateralization to the basilar tip. The left vertebral did communicate with the basilar but poorly through a small lumen and did not appear involved with the probable dissection (Figure 5). There was no anteroposterior flow through either PCOM. A 6F Cordis XB MPC guide catheter was placed into the right vertebral artery at the level of C2.

The aneurysm seemed to fill in two steps, with an initial irregular segment of the aneurysm being the site of an inflow jet extending laterally to the left followed by filling of a posteriorly prominent belly and final emptying anteriorly into the apparently intact distal basilar third (Figure 6). This shelf-like irregularity within the proximal aspect was most concerning in terms of the possible site of rupture (Figure 7) and most worrisome in terms of appearing to represent an area of transmural dissection. This area was also closest to the foramen of Luschka on the left, most likely the route of entry for the primarily intraventricular hemorrhage at presentation. At the upper more distal aspect of the aneruysm, there was a 90 degree tortuosity at the site of transition from the belly of the aneurysmal dilatation back into the normal basilar third anteriorly (Figure 1).

I elected to coil the aneurysmal dilatation without a balloon inside the stent which might have allowed visualization and protection of the stent lumen since removal of the balloon would likely cause rebound of the kinking of the artery, with unpredictable results. Traversal of the aneurysmal dilatation with a Prowler Select Plus microcatheter loaded with a Platinum Transend microwire necessitated traversal of the anerysmal belly with the microwire, prompting changing of the Transend microwire for a softer X-pedion 10 microwire. This microwire was advanced into the right PCA to the level of P2, straightening the segment of microwire within the aneurysmal segment and allowing

Figure 5

the traversal of the lesion with the Prowler Select Plus microcatheter without the need to subject the belly of the aneurysm to undue force. An Echelon-10 microcatheter was then steam-shaped with a 45 degree distal tip, loaded with a Platinum Transend microwire, and advanced into the distal dorsal lobe of the aneurysmal belly. Half of a 13 x 43 Presidio-18

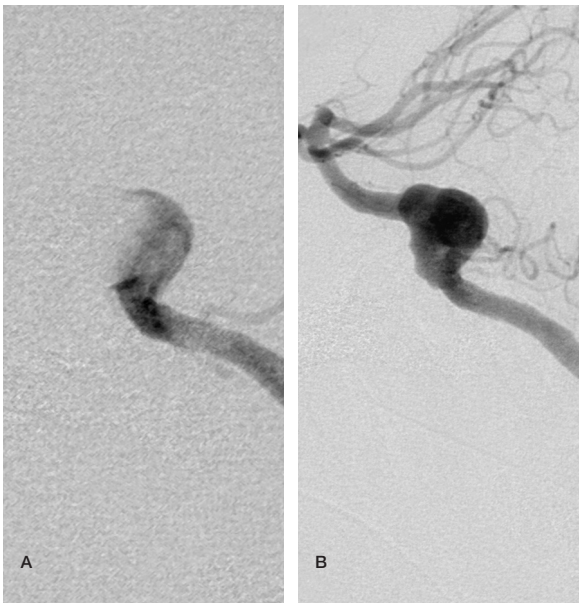

Figure 6

Figure 7

coil was then advanced into the belly, followed by deployment of the distal half of a 4.5 x 37mm Enterprise stent (Figure 8). Movement of the coil mass by this partial stent deployment gave me some confidence that the stent was displacing coil from the parent lumen and thus the rest of the stent was deployed, followed by deployment of the rest of the first coil. The aneurysm was then coiled with nine Cashmere-14 coils (12 x 30, 12 x 30, 12 x 19, 11 x 27, 11 x 18, 9 x 25, 9 x 22, 9 x 14, and 7 x 11), with the inferior irregularity (24 lateral 14) able to be successfully packed. Heparinization was increased as the dome was packed more and more tightly, and once the proximal portion was able to be packed, 300 mg Plavix and 325 x 2 ASA were given down an NG tube.

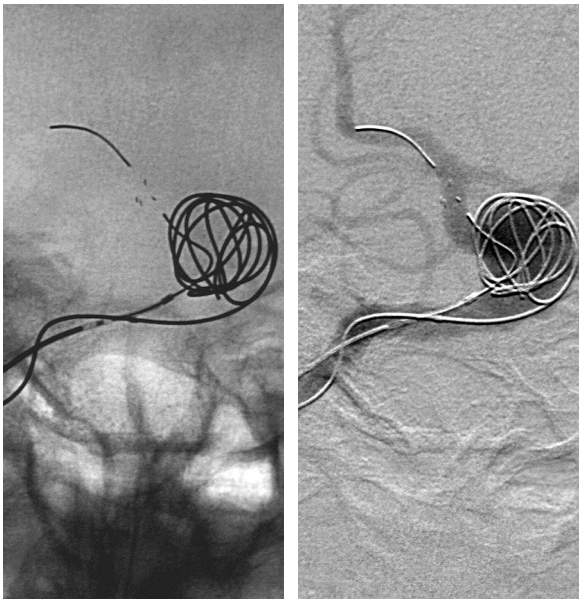

Figure 8

Postembolization angiography starting at 10:40 showed no sign of nontarget embolization or slowing of the transit time of contrast past the coil mass for approximately 30 minutes, and the decision to stop the case was made at 11:04. Multiple angiograms were obtained in an attempt to see any clot within the visualizable portions of the parent vessel and then the guide catheter was pulled back to the proximal right vertebral and angiography was performed to double check the vertebral for any sign of dissection or spasm at the site of the tip of the guide catheter. Slowing of the washout of contrast from the right vertebral was noted, despite a lack of a delay in the passage of contrast across the coil mass. Three cerebral angiograms were then performed, and suddenly there was a marked slowing of flow across the aneruysm (Figure 9) at 11:29, approximately 50 minutes after the detachment of the final coil.

The guide catheter was readvanced into a working position and the Echelon-10 microcatheter loaded with the X-pedion microwire were readvanced into position across the neck of the aneurysm, which was laced with 10 mg of ReoPro. Angiography immediately after this lacing, at 11:40 hours, 11 minutes after noticing the slowing, showed complete patency. This was the first angiogram I performed after crossing the stent with the microcatheter. Reocclusion of the basilar was then seen five minutes after the lacing with ReoPro. Re-traversal of the stent with the microcatheter was accompanied by a sudden release of contrast into the distal vasculature. This happened as the microcatheter crossed the portion of the parent vessel which appeared bent forward in a 90 degree shape on the 3D views of the aneruysm. This led to the thought that the occlusion was not due to clot but rather due to an accentuation of the bent portion of the parent vessel into a kink. Such an accentuation of tortuosity into a kink might have been caused by clot expanding the volume of the coiled portion of the

aneruysm. Accordingly, the Echelon-10 was exchanged for the Prowler Select Plus using a Transend floppy exchange wire, and a second Enterprise stent, this time a 4.5 x 28, was placed within the first (Figure 10). Three series of cerebral angiograms over 30 minutes showed no further tendency for the basilar to occlude and the guide catheter was then removed and the sheath stitched in.

Outcome

The patient was not able to move after awakening. The anesthesiologist thought this might have been due to a late administration of paralytic during the case. I brought the patient back down for angiography at 16:55, approximately four hours later, when he still had not recovered significant movement. A preprocedural head CT showed no significant change. The basilar was found to be widely patent. The patient regained his ability to move his arms and then his legs over two days. Follow-up angiography on 25th October showed that the coiling was stable and that apart from a small pocket at its upper aspect the aneurysm was occluded.

Discussion

I was unable to visualize the lumen directly and so my contention that the occlusion was caused by kinking and not by thrombosis of the lumen or coil-mediated crimping of the stent is essentially unprovable. Thrombosis of the lumen seems unlikely, as this would not be expected to cause re-occlusion more quickly, after only five minutes, after lacing the stent with 10 mg of ReoPro. Occlusion due to crimping of the stent lumen by coils should not have taken 50 minutes to express itself the first time but as a subocclusive crimping might also be worsened by thrombosis within the coiled portion of the aneurysm this is a possible explanation. I consider crimping by coils less likely than kinking at the site of tortuosity simply since I have not heard of crimping by coils during stent-assisted coiling in other hands but I might have packed more tightly and stent-assisted coiling of ruptured fusiform aneurysms is a relatively new approach. In any case, this explanation of coil-related crimping instead of tortuosity-related kinking would not obviate the essential conclusions of the case for me, as noted below.

Figure 9

Conclusions

The reconstructed parental vessel in this unsacrificable probable Mizutani 1 dissecting aneurysm was tortuous with a focal 90 degree bend. Although placement of a balloon within the stent lumen would simplify coiling and likely assist with the acquisition of a balloon axial

Figure 10

view, removal of the balloon would be accompanied by rebound of the tortuosity in an unpredictable fashion.

Washout of contrast from the upstream feeding artery proved more indicative of stenosis than flow of the leading edge of contrast through the mechanical restriction to flow.

I often watch an aneurysm with a wide neck for 30 minutes after embolization to make sure that no platelet aggregates form, especially on a wide neck or after stenting. Perhaps this time period should be increased to 60 minutes in cases where expansion of thrombus within a significant coiled volume could compromise a kinked or crimped stent. It was only due to my performing multiple angiograms after the placement of the last coil and my decision to check the extradural vertebral at the site of guide catheter placement for possible vasospasm that I noticed the initially very subtle slowing of flow 50 minutes after the detachment of the last coil and I did not see the frank slowing until approximately three angiograms later.

The benefit of a second stent in this case prompts me to recommend either the upfront placement of two stents initially before coiling, or reaquisition of the stent lumen with a microwire before coiling to facilitate easier placement of a second stent after coiling.

In the latter approach the wire should probably be a soft 10 microwire to minimize its support for the possibly kinked or crimped stent so as to allow any problem with flow to express itself.

References

1 Coert BA et Al: Surgical and endovascular management of symptomatic posterior circulation fusiform aneurysms. J Neurosurg 106: 855-865, 2007.
2 Flemming KD et Al: Prospective risk of hemorrhage in patients with vertebrobasilar nonsaccular intracranial aneruysm. J. Neurosurg 101: 82-87, 2004.
3 Berenstein A, Lasjaunias P, ter Brugge KG: Surgical Neuroangiography. Springer-Verlag, Berlin 2004.
4 Mizutani T et Al: Recurrent subarachnoid hemorrhage from untreated ruptured vertebrobasilar dissecting aneurysms. Neurosurgery 36: 905-913, 1995.
5 Yamaura A, Watanabe Y, Saeki N: Dissecting aneurysms of the intracranial vertebral artery. J Neurosurg 72: 183-188, 1990.
6 Sasaki O et Al: A clinicopathological study of dissecting aneurysms of the intracranial vertebral artery. J Neurosurg 75: 874-882, 1991.
7 Seifert V, Raabe A, Stolke D: Management-related morbidity and mortality in unselected aneurysms of the basilar trunk and vertebrobasilar junction. Acta Neurochir (Wien) 143: 343-349, 2001.
8 Gobin YP et Al: Treatment of large and giant fusiform intracranial aneurysms with Guglielmi detachable coils. J Neurosurg 84: 55-62, 1996.
9 Hoi Y et Al: Effects of arterial geometry on aneurysm growth: three-dimensional computational fluid dynamics study. J Neurosurg 101: 676-681, 2004.

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